THE ROYAL SOCIETY

doi 10.1098/rspb.2000.1248

Dynamics of a feline virus with two transmission modes within exponentially growing host populations

Kevin Berthier[1]*, Michel Langlais[2], Pierre Auger[1] and Dominique Pontier[1]

[1]*UMR CNRS 5558 'Biométrie et Biologie Évolutive', Université Claude Bernard Lyon 1, 43 Boulevard du 11 Novembre 1918, 69622 Villeurbanne Cedex, France*

[2]*UMR CNRS 5466 Mathématiques Appliquées de Bordeaux, UFR MI2S, BP 26, Université Victor Segalen Bordeaux 2, 146 Rue Léo-Saignat, 33076 Bordeaux Cedex, France*

Feline panleucopenia virus (FPLV) was introduced in 1977 on Marion Island (in the southern Indian Ocean) with the aim of eradicating the cat population and provoked a huge decrease in the host population within six years. The virus can be transmitted either directly through contacts between infected and healthy cats or indirectly between a healthy cat and the contaminated environment: a specific feature of the virus is its high rate of survival outside the host. In this paper, a model was designed in order to take these two modes of transmission into account. The results showed that a mass-action incidence assumption was more appropriate than a proportionate mixing one in describing the dynamics of direct transmission. Under certain conditions the virus was able to control the host population at a low density. The indirect transmission acted as a reservoir supplying the host population with a low but sufficient density of infected individuals which allowed the virus to persist. The dynamics of the infection were more affected by the demographic parameters of the healthy hosts than by the epidemiological ones. Thus, demographic parameters should be precisely measured in field studies in order to obtain accurate predictions. The predicted results of our model were in good agreement with observations.

Keywords: epidemiology; mathematical model; panleucopenia virus; domestic cat; *Felis catus*

1. INTRODUCTION

Determining the key variables responsible for microparasite persistence and the patterns of their subsequent endemicity is of crucial interest. In contrast to human infection diseases, knowledge of the infectious disease dynamics of wild mammalian populations is often quite fragmented (Gulland *et al*. 1993). Only a few parasites have been monitored in natural populations in sufficient detail for providing the necessary basis for testing the predictions of theoretical models. The most documented cases concern viruses characterized by direct transmission, such as the myxoma virus (Ross & Tittensor 1986; Trout *et al*. 1992), the phocine distemper virus (Dietz *et al*. 1989; Harwood 1990; Heide-Jørgensen *et al*. 1992) or the feline retroviruses (Courchamp *et al*. 1995; Fromont *et al*. 1997, 1998; Pontier *et al*. 1998).

Another case is feline panleucopenia virus (FPLV). This virus was introduced on Marion Island with the aim of eradicating the cat population, which had become a serious threat to seabirds (Van Rensburg *et al*. 1987). FPLV is characterized by a high virulence which mainly affects kittens (Csiza *et al*. 1971) and cats which recover from infection and develop an immune response which lasts several years (Scott & Geissinger 1997). The main novelty of FPLV is its high resistance outside the host, as it remains infectious for more than one year (Csiza *et al*. 1971). Transmission may thus occur either directly through contacts between an infected cat and a healthy cat or indirectly when a healthy cat sniffs the excrement of an infected cat. The relative implications of both modes of transmission in the persistence of the disease have not yet been analysed. One might expect that the high survival of the virus outside the host should enhance the likelihood of disease persistence.

Most current models have focused on directly transmitted viruses (Anderson & May 1991). Here, we developed a new model in order to understand the effect of the two modes of transmission on the host and virus dynamics. The model we used was deterministic and derived from the compartmental models of Anderson & May (1979). The distinguishing characteristic of our model lies in the coupling of the equations describing the direct transmission (susceptible, infected and recovered (SIR) part) to an equation driving the indirect transmission, i.e. the dynamics of the spatial contamination of the environment. The demographic data available on the cat population since the introduction of the virus on Marion Island enabled us to assess whether the model fitted the data adequately. A particularly important variable in epidemiological models is the function describing the pattern of efficient contact rates between individuals. The number of new infections per time-unit depends on the relationship between population size (measured as a number or a density) and contact behaviour for efficient disease transmission. It is traditionally assumed that efficient contact rates are either stable (proportionate mixing) (Hethcote & Yorke 1984) or linearly increasing (mass action) (Anderson & May 1979; May & Anderson 1979) with population density. As no information is currently available for FPLV, we compared the behaviours predicted by the model to observed data using two alternative forms, i.e. mass action and proportionate mixing, in order to determine which functional form is best adapted to describing the recruitment of new infected individuals. Ultimately, we conducted an elasticity analysis for determining which of the parameters of the model have the most influence on disease dynamics.

*Author for correspondence (berthier@biomserv.univ-lyon1.fr).

Received 10 February 2000 *Accepted* 12 July 2000

2. MATERIAL AND METHODS

(a) *The host population*

Marion Island is an island of 290 km^2 located in the southern Indian Ocean (46°52′ S, 37°51′ E). Five cats introduced in 1949 gave rise to a population estimated at 2137 individuals in 1975, 40% of the cats being less than one year old (Van Rensburg *et al*. 1987). The mean density was equal to nine cats per square kilometre (cats were present on 234 km^2). The cat population was estimated at 3400 in March 1977 when the virus was introduced. We thus assumed that the cat population demography was Malthusian until the introduction of the virus. The annual increase in the population was estimated at 23% per year (Van Aarde 1978).

In 1977, 96 cats were trapped, inoculated with FPLV by intraperitonal injection (Howell 1984) and then released (Van Aarde 1984). The mean density dropped down to four cats per square kilometre within two years (Van Aarde 1984). The population size was estimated at 615 cats in 1982 (Van Rensburg *et al*. 1987), corresponding to an annual decrease of 29%. From 1982, the annual decay rate slowed down to 8%. Antibodies against FPLV were detected in surviving cats, suggesting that panleucopenia was unable to eradicate the cat population. An important culling effort after 1982 caused the eradication of cats in 1991 (Bester 1993).

(b) *The virus*

FPLV is a member of the parvoviridae family that infects all Felidae and some other carnivore species (Reif 1976). It is a highly contagious cat viral disease characterized by severe gastroenteritis with fever, anorexia, vomiting, diarrhoea and marked leucopenia leading to the death of the animal (Legeay 1992). The incubation period is very short (one to four days) (Legeay 1988) and infection lasts for two weeks. Infected cats are viremic for a few days and excrete the virus in their faeces, urine, saliva, vomitus and nasal and ocular discharges (Csiza *et al*. 1971; Reif 1976).

Susceptible cats are infected by either direct contact with an infected carrier or by mechanical transmission via infected premises. The virus is resistant to physical conditions (Riser 1943) and may survive on infected premises for one year (Mac Pherson 1956). Receptivity does not differ between sexes (Reif 1976) but kittens are more susceptible (80% of mortality) than adults (20% of mortality) (Legeay 1988). Infected cats that recover from infection develop an immunity for at least six years (Scott & Geissinger 1997), do not become excreting carriers and have a normal life expectancy. Given the life expectancy of cats in natural populations (four to six years) (Pontier 1993), we considered that immunity to the disease is lifelong. Immune females have a normal birth rate and kittens from immune females become susceptible when maternally derived antibody wanes at approximately ten weeks (Reif 1976).

(c) *The model of FPLV propagation*

The first part of the model was built on the framework of Anderson & May (1979, 1991). Let N be the density of cats at time t. The natality rate (b) and death rate (m) are constant, with a population growth rate of $r = b - m > 0$. In the absence of the parasite, the population grows exponentially (Van Aarde 1984):

$$\frac{dN}{dt} = rN. \tag{1}$$

Once the virus is introduced, the population splits into three classes: susceptible (having density S), infectious (having density I) and recovered or immune (having density R) individuals. We assumed that all females, whatever their health status, have a normal birth rate and that their kittens become rapidly susceptible. Susceptible and immune individuals are assumed to die at a constant per-capita death rate m. Infected individuals suffer an additional risk of mortality due to infection. Let $\alpha > 0$ be the inverse of the length of the infectious period and let e be the proportion of infected cats subsequently recovering from the disease ($0 < e < 1$). We assumed that a cat that has recovered from infection is immune to reinfection for the rest of its life. The dynamics of FPLV within a population of cats is governed by the following set of differential equations (figure 1):

$$\frac{dS}{dt} = b(S + I + R) - mS - \sigma(S, I, R), \tag{2}$$

$$\frac{dI}{dt} = -(m + \alpha) + \sigma(S, I, R) \tag{3}$$

and

$$\frac{dR}{dt} = -mR + e\alpha I. \tag{4}$$

Two forms of incidence function are considered: (i) $\sigma(S, I, R) = \sigma_{ma}SI$ for the mass action model, where $\sigma_{ma} > 0$, or (ii) $\sigma(S, I, R) = \sigma_{pm}SI/(P)$ for the proportionate mixing model, where $\sigma_{pm} > 0$ and P is the total population density ($P = S + I + R$). This yields the model for direct transmission of the virus between an infected host and a susceptible one.

The second part of the model incorporates indirect transmission between a susceptible host and the contaminated environment. Cats mark their territory by spraying urine and leaving faeces at visually conspicuous sites. If detected, both urine and faeces are sniffed by both male and female cats (Bradshaw 1992). Here we assumed that susceptible cats are infected with contaminated faeces (because only a few viruses are contained in urine) (Pedersen 1988).

The area contaminated by one infectious cat per unit of time is equal to the number of faeces left by an infectious cat per unit of time (f) multiplied by the surface area of one faeces (β). On the other hand, infected areas of the environment are decontaminated at a rate d, with $1/d$ being the life expectancy of the virus outside the host. Let $H(t)$ be the surface of the environment contaminated by the virus at time t and let H_∞ be the total area where cats live on Marion Island. Then the dynamics of the environment contamination–decontamination are described by

$$\frac{dH}{dt} = \beta f I(t)(H_\infty - H(t)) - dH(t). \tag{5}$$

Set

$$C(t) = \frac{H(t)}{H_\infty}, \tag{6}$$

$$\phi = \beta f. \tag{7}$$

Equation (5) then becomes

$$\frac{dC}{dt} = \phi(1 - C)I - dC. \tag{8}$$

The full model, which is denoted as susceptible, infected, recovered and contaminated (SIRC) (figure 1), was obtained

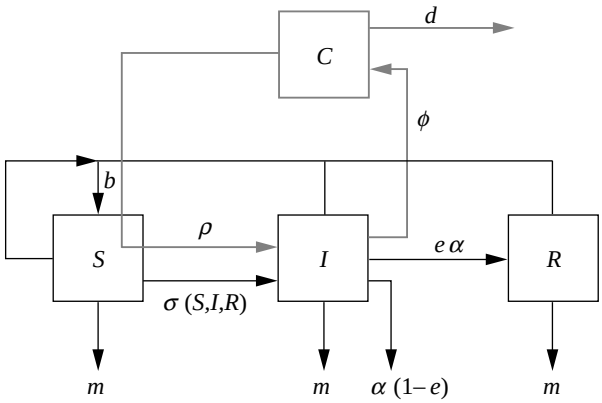

Figure 1. Compartmental representation of the SIRC model of a feral cat population of an island infected by FPLV. The epidemiological part of the model is in black and the contamination of the area studied part is in grey. Susceptible cats are denoted S, infectious cats are denoted I and immune cats are denoted R. C represents the proportion of the surface area studied contaminated by the virus. The arrows represent the passage rates between the different compartments. b is the birth rate, m is the natural death rate, α is the output rate of the infectious compartment, e is the proportion of the population which survives the disease, ϕ is the proportion of the island surface occupied by a cat territory and d is the natural rate of decontamination of the area studied infected by the virus. σ and ρ are the direct and indirect rates of transmission of the disease, respectively.

by coupling together equations (2)–(4), which describe direct transmission and equation (8), which describes indirect transmission. The rate of indirect transmission of the disease allows coupling of both parts. Let ρ be the rate at which each contaminated faeces leads to transmission of the disease when a cat sniffs it. The force of infection is

$$\rho\frac{H(t)}{H_\infty} \Leftrightarrow \rho C(t). \tag{9}$$

The dynamics of FPLV within the population of cats can be written as a set of four coupled, nonlinear, ordinary differential equations:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b(S+I+R) - mS - \sigma(S,\,I,\,R) - \rho CS, \tag{10}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = -(m+\alpha)I + \sigma(S,\,I,\,R) + \rho CS, \tag{11}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = -mR + e\alpha I, \tag{12}$$

and

$$\frac{\mathrm{d}C}{\mathrm{d}t} = \phi(1-C)I - dC. \tag{13}$$

The equation for the total density (P) reads

$$\frac{\mathrm{d}P}{\mathrm{d}t} = rP - (I-e)\alpha I. \tag{14}$$

It follows from equation (14) that, for $r > (1-e)\alpha$, the density is still experiencing a Malthusian growth and the virus cannot control the cat population. From now on we shall assume that

$$0 < r = b - m < (1-e)\alpha. \tag{15}$$

It follows from equation (15) that the natality rate (b) is smaller than the overall death rate $(m+(1-e)\alpha)$ of infectious individuals.

(d) *Elasticity analysis*

In order to determine the set of parameters that have more influence on disease dynamics, we conducted a series of numerical simulations analysing the elasticity of the model results, i.e. the equilibrium population size (P) and equilibrium prevalence (i^*). Note that the equilibrium prevalence was known from equation (14), i.e. $i^* = b - m/(1-e)\alpha$, so that the analysis for i^* could have been done analytically as well. This is also important because our model was based on parameter values estimated from available data and we had more confidence in the values of some parameters than others. The interval of variation was arbitrarily set at 10%. Let θ be one of the parameters of the model. We calculated $e_\theta = \theta\Delta P/P\Delta\theta$ and $e_\theta = \theta\Delta i^*/i^*\Delta\theta$, where $\Delta\theta = \tilde{\theta} - \theta$ with $\tilde{\theta} = \theta \pm 10\%\theta$.

3. RESULTS

(a) *Equilibrium and stability analysis*

(i) *Mass action SIRC*

There are two possible equilibria: destruction of the cat population or emergence of an endemic state (see Appendix A for details). The equilibrium $(S_1^* = I_1^* = R_1^* = C_1^* = 0)$ corresponding to the extinction of the host population is unstable because $r > 0$ and the virus cannot yield destruction of the host population. When both equation (15) and

$$1 < \frac{b}{m} < \frac{m+\alpha}{m+e\alpha} \tag{16}$$

hold, there is a unique equilibrium $(S_2^*,\, I_2^*,\, R_2^*,\, C_2^*)$ with positive entries and $0 < C_2^* < 1$, which cannot be expressed in close form except for the asymptotic prevalence deduced from equation (14) and given by $i_2^* = b - m/(1-e)\alpha$. Numerical simulations suggest that it is locally stable as soon as both equations (15) and (16) hold. Thus, from a numerical point of view these two conditions seem to be sufficient for the virus to control the host population. For large values of either ρ or σ the virus controls the cat population at very low densities. When equations (15) or (16) are not satisfied then the host population resumes a Malthusian growth.

(ii) *Proportionate mixing SIRC*

Still assuming equation (15) holds, besides the equilibrium $(S_1^* = I_1^* = R_1^* = C_1^* = 0)$, a second one $(S_2^*,\, I_2^*,\, R_2^*,\, C_2^*)$ with positive entries and $0 < C_2^* < 1$ can exist provided some extra and rather involved inequalities (not developed here) hold. It can be expressed in a close but complicated form with an asymptotic prevalence still given by $i_2^* = b - m/(1-e)\alpha$. Numerical simulations first suggest that this endemic state is locally stable when it exists. For large ρ and medium ranges of σ, the cat population is controlled at very low density. Next, when this endemic state does not exist (0, 0, 0, 0) can be either locally stable or not. Thus, both extinction and a Malthusian growth of the cat population can occur basically depending on the respective size of σ and ρ (other parameters lying in the range of field data), e.g. for large σ

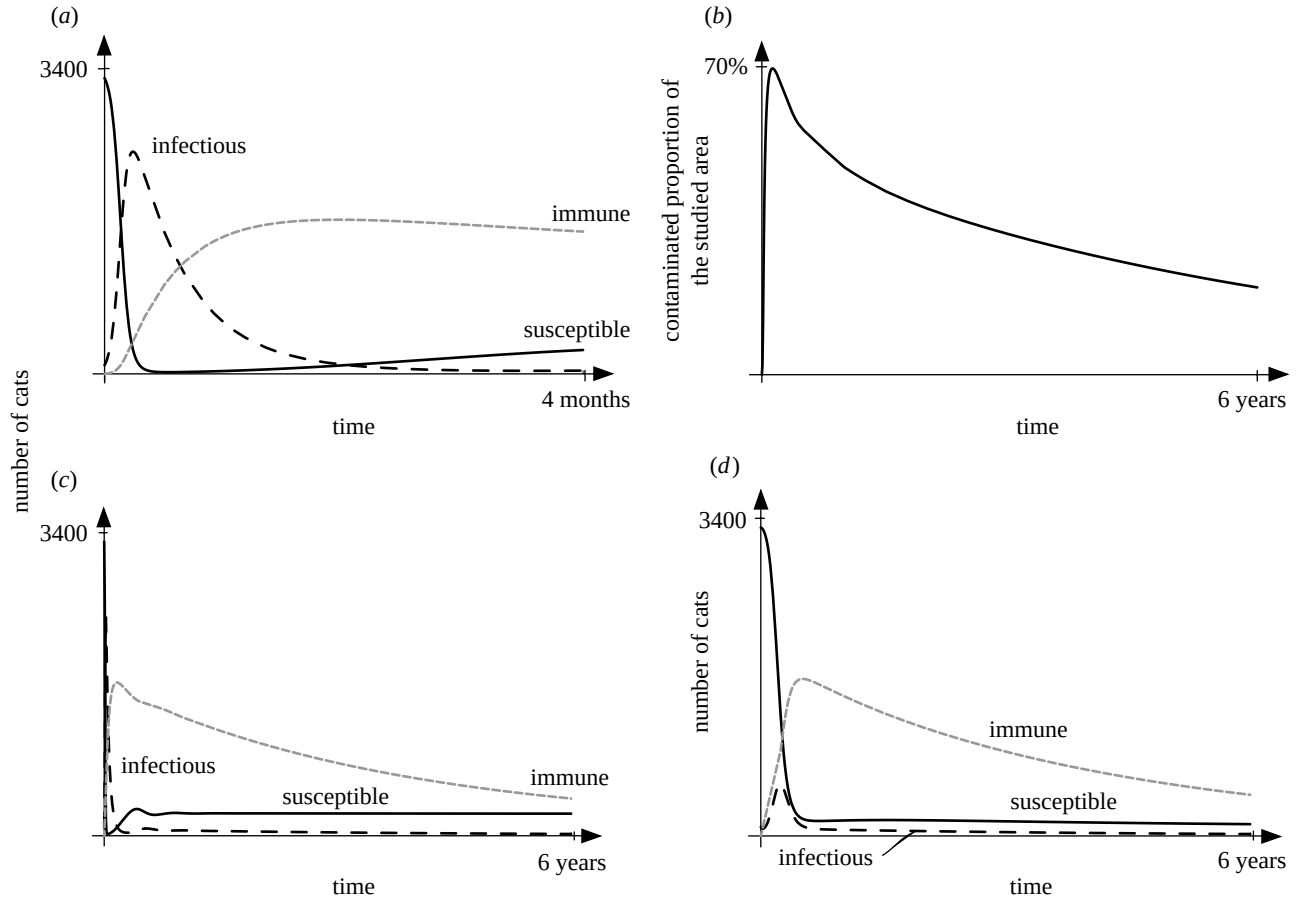

Figure 2. Simulations of the dynamics of the feral cat population on Marion Island after the introduction of FPLV. (*a–c*) SIRC model with mass action for the direct route of contamination. The values for the parameters are $b=1.10$, $m=0.89$, $\sigma=0.1$, $\rho=0.8$, $\alpha=26$, $e=0.55$, $\phi=0.01$ and $d=1$. All simulations began with 3300 susceptible (in black), 96 infectious (dashed) and no immune cats (in grey). (*a*) Zoom on the epidemic peak at the beginning of the infection. (*b*) Evolution of the proportion of the contaminated area for the first six years. (*c*) Depression of the population during the same period was the same as the monitoring in the field on Marion Island. (*d*) SIRC model with proportionate mixing. The initial conditions are the same as those of the mass action SIRC. The same parameters are used except for $\sigma=10$ and $\rho=20$. It allows a weak epidemic peak like the one observed on Marion Island.

extinction occurs and for small σ and ρ Malthusian growth occurs.

(iii) *Indirect transmission alone*

When direct transmission is ignored, i.e. $\sigma(S, I, R)=0$, then the host population resumes a Malthusian growth for small ρ, while for large ρ an endemic state (S_2^*, I_2^*, R_2^*, C_2^*) with positive entries and $0 < C_2^* < 1$ can exist and FPLV controls the host population.

(b) *Application to biological data*

We used data from Marion Island or, when lacking thereof, from other subantarctic cat populations (Kerguelen Islands) which were very similar in terms of the number of founder cats, host population dynamics, diet and climatic conditions. The area unit was the surface occupied by cats on Marion Island, i.e. 234 km^2. All parameters were estimated per year. Natality (b) was estimated at 1.10 per cat per year (Kerguelen Islands) (Pascal 1980). Based on the annual growth rate of the population before the introduction of the virus ($r=23.3\%$), it was possible to estimate the natural mortality rate as $m=0.89$ per cat per year. Mortality due to the disease was strongly dependent on age (20% of infected adults and 20% of infected kittens died from the disease) (Legeay 1988). Approximately 40% of the cat population was less than one year old (Van Rensburg *et al.* 1987). Once infected a cat either died or recovered from the disease after two weeks. Thus, we assumed that infected individuals left the infectious compartment at a rate $\alpha=26$ (α is the inverse of the duration of the disease in a year, which was set at two weeks). The proportion of cats which recovered was $e=0.56$ (i.e. $0.40\times0.20+0.60\times0.80$). The virus survives one year outside the host, thus $d=1$. Cats shed their faeces at visually conspicuous sites in order to mark their territory. We assumed that the territory of an infected cat was contaminated over its whole surface. We used values of home range size estimated in similar conditions (Liberg *et al.* 2000), i.e. 2.5 km^2. Thus, we took $\phi=0.01$ ($=2.5/234$). The last two parameters of the model were somewhat unknown, i.e. σ and ρ.

(c) *Simulations*

The densities of susceptible, infected and immune individuals are plotted against time in figure 2.

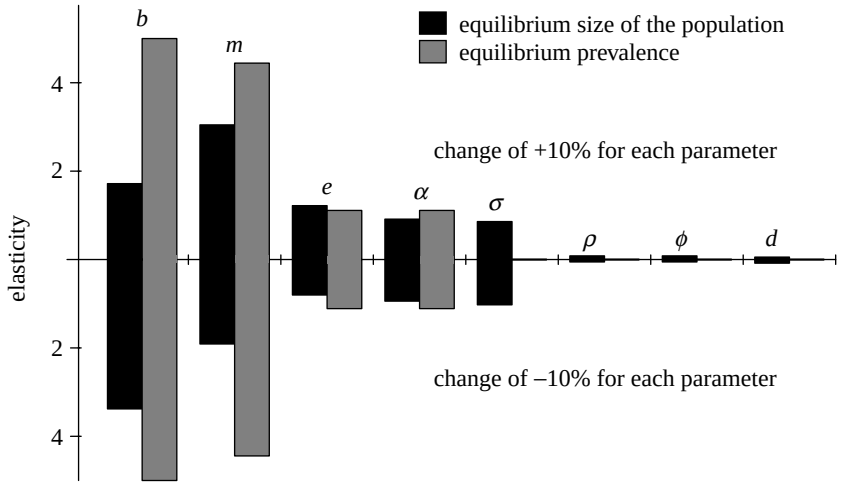

Figure 3. Elasticity of the SIRC model with a mass-action incidence term for the direct mode of transmission of the disease (based on the values of the parameters and initial conditions used in figure 2*a*). Variation of the two output parameters as a function of each of the entry parameters. The two output parameters are the number of cats at equilibrium and the prevalence of the disease at equilibrium.

(i) *Mass action SIRC*

We selected $\sigma = 0.1$, which was adapted from Fromont *et al.* (1998) and increased due to its numerous routes of direct transmission. By varying ρ from 0.1 to 80, we observed that infected individuals increased rapidly within the first month, thereby spreading the virus through the whole population (figure 2*a*). The proportion of the total area contaminated by the virus increased strongly within the first year and slowly decreased thereafter (figure 2*b*). The population size at the equilibrium state was much lower than it would have been in the absence of infection, indicating that the virus was able to control the cat population. For $\rho = 0.8$, the total number of cats decreased from 3400 to 685 after six years (figure 2*c*), which is very close to observed field data (versus 615 ± 107) (Van Rensburg *et al.* 1987). The output of the model compared favourably with field data with respect to the course of the population size variation during the first six years following introduction of the virus (Van Rensburg *et al.* 1987). Numerical simulations showed that small variations in ρ and σ did not alter the transient dynamics drastically.

(ii) *Proportionate mixing SIRC*

We selected $\sigma = 10$, which was again adapted from Fromont *et al.* (1998). Now, the cat population increased for small ρ. Increasing ρ led to control of the host population at acceptable densities, but recovering the observed transient dynamics required an unacceptable indirect transmission rate ($\rho = 20$). Small variations in ρ and σ did not alter this phenomenon.

At times of large σ and ρ there was no significant difference between the dynamics driven by the two incidence terms for direct transmission.

(iii) *Indirect transmission alone*

Numerical simulations showed dynamics similar to the previous case of proportionate mixing incidence.

(d) *Elasticity analysis for mass action SIRC*

Figure 3 shows the results of this analysis. The parameters which most affected the population size at equilibrium were the natural mortality rate (m) and birth rate (b), with the survival rate of the disease (e), length of the disease period (α) and transmission efficiency (σ) of the direct route of transmission having a much lesser effect. From its explicit expression, as derived from equation (14), the equilibrium prevalence was extremely sensitive to m and b and, to a much lesser degree, to e and α. Summing up, FPLV will mainly be driven by the demographic parameters of the cat population and less importantly so by the characteristics of the infection (e and α). The impact of the infection on the host population size was also much more sensitive to the direct than indirect transmission mode of the infection.

4. DISCUSSION

To our knowledge, the model presented here is the first one devoted to parvovirus and provides an insight into understanding FPLV dynamics in domestic cat populations. The specificity of the host–parasite system modelled here lies in the two modes of propagation of the virus, i.e. through either direct contact between infectious and susceptible cats or indirect transmission between a healthy cat and the contaminated environment. The key parameter of the indirect transmission is ϕ which drives the dynamics of the surface of the area contaminated by the virus from the dynamic process of contamination–decontamination of individual home ranges. The social and spatial organization of cat populations is highly variable (Liberg *et al.* 2000) as is the population dynamics (Pontier 1993). One needs to bear in mind that the model has been applied to the particular case of a host population growing exponentially on a delimited area where individuals live in large home ranges.

Although the model is relatively simple, it was possible to describe the dynamics of the epidemic disease accurately for the six years following the introduction of the virus to Marion Island. First, our simulation results showed that mass action was more satisfactory than proportionate mixing in describing the direct transmission dynamics on Marion Island. Second, because

domestic cats had been eradicated by other means six years after the introduction of the virus to the island (Bester 1993), it was not clear how the FPLV–cat system would have evolved naturally in the long term. When equations (15) and (16) were fulfilled our model predicted that the infection was maintained, leading to a stable equilibrium. The host population decrease was huge (95% six years after the introduction of the virus, compared to a population free of FPLV) and the results showed that the virus was able to maintain the hosts at a low size (600 versus 3400 cats before introduction of FPLV). The predicted proportion of immune cats was *ca.* 29%, which falls within the range of values observed in continental cat populations (Fromont *et al*. 1996). Third, the elasticity analysis allowed us to identify the most influential parameters in our model. The results showed that the epidemic dynamics were mainly driven by the demographic parameters of the healthy cats. Note, however, that, in natural conditions, parameters have different variability so their influence on the behaviour on the natural system was not fully reflected by the sensitivity analysis of the model. A precise measure of these parameters in field studies is thus required. Simulations suggested that indirect transmission acts as a reservoir that supplies the host population with a few infected individuals but in sufficient numbers to permit the virus to spread rapidly to the whole population through direct contacts.

Modelling domestic cat populations with different demographic schedules, i.e. logistic growth, as observed in most continental populations (Pontier 1993), is needed in order to complete the results of the present study. These results are not easy to extrapolate to other cat populations exhibiting different demographic and social structures. A single indirect transmission can control the host population but this was ruled out here only because the transient behaviour did not fit the data. The same conclusion can be drawn for a direct incidence of the proportionate mixing type. Note that only this transmission mode can lead to the extinction of the host population. It was observed that more than 90% of individuals were immune in an urban cat population (Fromont *et al*. 1996). The persistence of the virus in such conditions is probably greatly enhanced by its capacity for retaining infectivity in the environment for at least one year. Thus, when contacts between cats are low or when the proportion of immune cats is very high, the virus can persist between new generations of susceptible cats. This expectation is strengthened by the particular seasonal epidemiological pattern observed in most continental cat populations: a predominance of cases appears during summer and mostly corresponds to juveniles (e.g. Reif 1976).

Keeping in mind that Marion Island may represent a typical model for a subantartic ecosystem where cats were introduced, one might conclude that the parvovirus is unable to eradicate such exponentially growing cat populations. This result is consistent with the fact that the antibody titres in the cat population increased after the introduction of the virus (Van Rensburg *et al*. 1987) which led to the decision of eradicating cats in other ways (culling operations) (Bloomer & Bester 1991). However, maintaining a pest species at a low size might be a better strategy than eradication, depending on the situation (Courchamp *et al*. 1999). This is the case when a predator (cat) and its prey (rabbits and rodents) are simultaneously introduced to large islands (Courchamp *et al*. 1999), such as the Kerguelen Islands, where population elimination is much more difficult. Rabbits and rats might do more harm than cats, in which case control of cats might be preferable than eradication (Cleaveland *et al*. 1999; Courchamp *et al*. 1999). On the other hand, if FPLV is a good candidate with regard to its ability to control island cat populations, the lack of specificity of the virus enhances the risk of establishment in non-target species (canids and felids) (Johnson 1964; Spencer 1991). One must be highly cautious before using this pathogen as an agent for biological control on oceanic islands.

We thank N. Bahi-Jaber, F. Courchamp, E. Fromont and J. M. Gaillard for helpful comments. This work was supported by the French Polar Institute (IFRTP programme no. 279) and Centre National de la Recherche Scientifique (programme 'Modélisation de la Circulation de Parasites dans des Populations Structurées').

APPENDIX A. MATHEMATICAL ANALYSIS OF THE MASS-ACTION SIRC MODEL

The cat–FPLV model with a mass-action incidence function reads as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = b(S+I+R) - mS - \sigma_{\mathrm{ma}}SI - \rho CS, \tag{A1}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = -(m+\alpha)I + \sigma_{\mathrm{ma}}SI + \rho CS, \tag{A2}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = -mR + e\alpha I \tag{A3}$$

and

$$\frac{\mathrm{d}C}{\mathrm{d}t} = \phi(1-C)I - dC. \tag{A4}$$

(a) *Elementary stability analysis*

We searched for constant solutions (S^*, I^*, R^*, C^*) of equations (A1)–(A4). S^*, R^* and I^* can each be expressed as a function of C^*:

$$S^* = \frac{\alpha + m - b\left(1 + \dfrac{e\alpha}{m}\right)}{(b-m)} I^*, \tag{A5}$$

$$R^* = \frac{e\alpha}{m} I^* \tag{A6}$$

and

$$I^* = \frac{d}{\phi} \frac{C^*}{(1-C^*)}. \tag{A7}$$

Now we substitute these relationships into equation (A1) at equilibrium and find

$$(1 - \sigma\Delta I^* - \rho\Delta C^*), \tag{A8}$$

with

$$\Delta = \frac{\alpha + m - b\left(1 + \dfrac{e\alpha}{m}\right)}{(b-m)(m+\alpha)}. \tag{A9}$$

A first solution of equation (A8) is $I^* = 0$, which yields $C^* = R^* = S^* = 0$. Next, there is a unique positive solution (C^*) of equation (A8) with $0 < C^* < 1$ when the numerator of Δ is positive; this is checked from a graphical analysis of

$$F(C^*) = \sigma\Delta\frac{d}{\rho}\frac{C^*}{(1-C^*)} + \rho\Delta C^* = 1. \tag{A10}$$

The positivity Δ is equivalent to

$$1 < \frac{b}{m} - \frac{m+\alpha}{m+e\alpha}. \tag{A11}$$

The Jacobian of the mass action SIRC system is

$$\mathcal{J}_{(S,I,R,C)} = \begin{bmatrix} b-m-\sigma I-\rho C & b-\sigma S & b & -\rho S \\ \sigma I+\rho C & \sigma S-(m+\alpha) & 0 & \rho S \\ 0 & e\alpha & -m & 0 \\ 0 & \phi(1-C) & 0 & -\phi I-d \end{bmatrix}. \tag{A12}$$

Evaluated at the equilibrium $(0, 0, 0, 0)$ one has

$$\mathcal{J}_{(0,0,0,0)} = \begin{bmatrix} b-m & b & b & 0 \\ 0 & -(m-\alpha) & 0 & 0 \\ 0 & e\alpha & -m & 0 \\ 0 & \phi & 0 & -d \end{bmatrix}, \tag{A13}$$

so that the trivial equilibrium is unstable because $b > m$.

There is no simple formulation of the second implicit stationary state, but numerically it seems to be always stable with realistic values of the parameters of the model.

REFERENCES

Anderson, R. M. & May, R. M. 1979 Population biology of infectious diseases. Part I. *Nature* **280**, 361–367.

Anderson, R. M. & May, R. M. 1991 *Infectious diseases of humans: dynamics and control*. Oxford University Press.

Bester, M. N. 1993 Eradication of cats from Sub-antarctic Marion Island. In *Proceedings of the Sixth International Theoretical Congress* (ed. M. L. Augee), p. 24. Sydney: University of New South Wales.

Bloomer, J. P. & Bester, M. N. 1991 Effects of hunting on the population characteristics of feral cats on Marion Island. *S. Afr. J. Wildl. Res.* **21**, 97–102.

Bradshaw, J. W. S. 1992 *The behaviour of the domestic cat*. Wallingford, UK: CAB International.

Cleaveland, S., Thirgood, S. & Laurenson, K. 1999 Pathogens as allies in island conservation. *Trends Ecol. Evol.* **14**, 83–84.

Courchamp, F., Pontier, D., Langlais, M. & Artois, M. 1995 Population dynamics of feline immunodeficiency virus within populations of cats. *J. Theor. Biol.* **175**, 553–560.

Courchamp, F., Langlais, M. & Sugihara, G. 1999 Cats protecting birds: modelling the mesopredator release effect. *J. Anim. Ecol.* **68**, 282–289.

Csiza, C. K., Scott, F. W., De Lahunta, A. & Gillespie, J. H. 1971 Immune carrier state of feline panleucopenia virus-infected cats. *Am. J. Vet. Res.* **32**, 419–426.

Dietz, R., Heide-Jørgensen, M. P. & Härkönen, T. 1989 Mass death of harbours seals *Phoca vitulina* in Europe. *Ambio* **18**, 258–264.

Fromont, E., Artois, M. & Pontier, D. 1996 Cat population structure and circulation of feline viruses. *Acta Oecol.* **17**, 609–620.

Fromont, E., Artois, M., Langlais, M., Courchamp, F. & Pontier, D. 1997 Modelling the feline leukemia virus (FeLV) in natural populations of cats (*Felis catus*). *Theor. Pop. Biol.* **52**, 60–70.

Fromont, E., Pontier, D. & Langlais, M. 1998 Dynamics of a feline retrovirus (FeLV) in host populations with variable structure. *Proc. R. Soc. Lond.* B **265**, 1097–1104.

Gulland, F. M. D., Albon, S. D., Pemberton, J. M., Moorcroft, P. R. & Clutton-Brock, T. H. 1993 Parasite-associated polymorphism in a cyclic ungulate population. *Proc. R. Soc. Lond.* B **250**, 7–13.

Harwood, J. 1990 A short history of the North Sea seal epidemic. *Ocean Challenge* **1**, 4–8.

Heide-Jørgensen, M. P., Härkönen, T., Dietz, R. & Thompson, P. M. 1992 Retrospective of the 1988 European seal epizootic. *Dis. Aquat. Org.* **13**, 37–62.

Hethcote, H. W. & Yorke, J. A. 1984 Gonorrhea transmission dynamics and control. *Lect. Notes Biomath.* **56**.

Howell, P. G. 1984 An evaluation of the biological control of the feral cat *Felis catus* (Linnaeus, 1758). *Acta Zool. Fenn.* **172**, 111–113.

Johnson, R. H. 1964 Isolation of a virus from a condition simulating feline panleucopenia in a leopard. *Vet. Rec.* **76**, 1008–1013.

Legeay, Y. 1988 Le point sur les maladies infectieuses félines. ENV Nantes. *J. Nantaises Pathol. Féline* 1–14.

Legeay, Y. 1992 Leucopénie infectieuse féline. *Encycl. Vét. Méd. Gén.* **1600** 1–5.

Liberg, O., Sandell, M., Pontier, D. & Natoli, E. 2000 Density, spatial organisation and reproductive tactics in the domestic cat and other felids. In *The domestic cat: the biology of its behaviour*, 2nd edn (ed. D. C. Turner & P. Bateson), pp. 119–148. Cambridge University Press.

Mac Pherson, L. W. 1956 Feline enteritis virus—its transmission to mink under natural and experimental conditions. *Can. J. Comp. Med. Vet. Sci.* **20**, 197–202.

May, R. M. & Anderson, R. M. 1979 Population biology of infectious diseases. Part II. *Nature* **280**, 455–461.

Pascal, M. 1980 Structure et dynamique de la population de chats harets de l'archipel des Kerguelen. *Mammalia* **44**, 161–182.

Pedersen, N. C. 1988 Feline panleucopenia. In *Feline infectious diseases* (ed. N. C. Perdersen), pp. 15–20. American Veterinary Publications.

Pontier, D. 1993 Analyse des traits d'histoire de vie chez les mammifères. In *Mémoire d'habilitation à diriger les recherches*. France: Université Lyon I.

Pontier, D., Fromont, E., Courchamp, F., Artois, M. & Yoccoz, N. G. 1998 Retroviruses and sexual dimorphism in domestic cat populations (*Felis catus* L.). *Proc. R. Soc. Lond.* B **265**, 167–173.

Reif, J. S. 1976 Seasonality, natality and herd immunity in feline panleucopenia. *Am. J. Epidemiol.* **103**, 81–87.

Riser, W. H. 1943 Infectious panleucopenia of cats. *N. Am. Vet.* **24**, 293–298.

Ross, J. & Tittensor, A. M. 1986 The establishment and spread of myxomatosis and its effects on rabbits populations. *Phil. Trans. R. Soc. Lond.* B **314**, 599–606.

Scott, F. W. & Geissinger, C. 1997 Duration of immunity in cats vaccinated with an inactivated feline panleucopenia, herpesvirus and calcivirus vaccine. *Feline Pract.* **25**, 12–19.

Spencer, J. A. 1991 Survey of antibodies to feline viruses in free-ranging lions. *S. Afr. J. Wildl. Res.* **21**, 59–61.

Trout, R. C., Ross, J., Tittensor, A. M. & Fox, A. P. 1992 The effect on a British wild rabbit population (*Oryctolagus cuniculus*) of manipulating myxomatosis. *J. Appl. Ecol.* **24**, 63–73.

Van Aarde, R. J. 1978 Reproduction and population ecology in the feral house cat, *Felis catus*, on Marion Island. *Carniv. Genet. Newslett.* **3**, 288–316.

Van Aarde, R. J. 1984 Population biology and the control of feral cats on Marion Island. *Acta Zool. Fenn.* **172**, 107–110.

Van Rensburg, P. J. J., Skinner, J. D. & Van Aarde, R. J. 1987 Effects of feline panleucopenia on the population characteristics of feral cats on Marion Island. *J. Appl. Ecol.* **24**, 63–73.

As this paper exceeds the maximum length normally permitted, the authors have agreed to contribute to production costs.